In that type of chronic nephritis characterized by marked albuminuria, cylindruria and edema, there were similar findings. In that type of chronic nephritis associated with hypertension, the non-protein nitrogen was increased, ranging from 40 to 181 milligrams per 100 c.c., and the percentage of the ammonia-urea fraction was usually higher than in non-nephritic cases. The nitrogen values in these patients were subject to rapid fluctuations in the course of a few days and clinical improvement was associated with a fall in the non-protein nitrogen content. Uremia was almost always accompanied by some increase of the non-protein nitrogen in the blood but no constant relation could be established between the degree of the increase and the tendency to uremia.

We believe that this method of estimating the total non-protein nitrogen in the blood is a valuable aid in the clinical study of nephritis and that it can be carried out in any thoroughly equipped clinical laboratory. The error of the method is indicated by the duplicate analyses which were done in almost all cases and which showed an average discrepancy between duplicates of 1.6 milligrams per 100 c.c. of blood. The urea method was in our hands less reliable, and large and inexplicable discrepancies occurred at times in our urea duplicates rendering repetition necessary and causing us to attach less importance to the urea figures.

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The toxicity of sodium tartrate with special reference to diet and tolerance.

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The toxicity of the sodium salts of dextro and levo tartaric acid was tested in experiments on frogs and rabbits. Both isomers were found equally toxic in these animals thus contradicting the earlier work of Chabrié[1] on the subject, who claimed that levo was more than twice as toxic as dextro tartaric acid. In experiments on rabbits, diet proved to be an important factor in the determination of resistance to this substance. Animals which were fed oats or oats

[1] *Compt. Rend. Acad. Sc.*, 1893, Vol. 116, p. 1410.

and cabbage succumbed to a dose of 0.4 gm. of the salt per kilo when given by subcutaneous injection. Suppression of urine was usually observed on the first day and death occurred in six to seven days. In starvation, slightly smaller doses were fatal to some rabbits. The resistance was increased considerably when the diet was changed to carrots. Such animals stood 1.0 gm. per kilo by subcutaneous injection, while 1.2–1.5 gm. per kilo were toxic. A moderate degree of tolerance for tartrates was induced in animals which were fed oats and cabbage. By gradually increasing the dose, a large proportion (6 out of 9) of rabbits survived 0.8 gm. per kilo which is twice the fatal dose. Rabbits which were receiving carrots did not acquire tolerance for tartrates. Sodium tartrate was much less toxic when given by mouth. 5 gm. per kilo was found to be the minimum fatal dose.

EXPERIMENTS ON CATS.

Amounts which have been found to be fatal for rabbits did not produce any symptoms in cats. Subcutaneous injection of one gm. per kilo produced a slight diarrhea in some individuals, and had no effect whatever in others. 1½ gm. per kilo proved fatal to one cat but was without action in another. Out of four cats which received 2 gm. per kilo three died, one survived. When sodium tartrate was given by mouth vomiting frequently occurred. In one case, however, when ten gm. per kilo were fed diarrhea was the only effect observed.

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The influence of pancreatic and duodenal extracts on the glycosuria and the respiratory metabolism of depancreatized dogs.

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Several dogs completely depancreatized by Hedon's method and eliminating glucose and nitrogen in Minkowski's ratio were treated by intravenous injection of pancreatic extract prepared by Knowlton and Starling's method.[1] The urines collected in twenty-four hour periods exhibited an increase in the D: N ratio

[1] Knowlton and Starling, *Journ. of Physiol.*, 1912, XLV, p. 146.